Coronary Artery Disease (IHD)

- Can lead to Myocardial ischemia, which is characterized by Angina Pectoris

Vasospastic (Prinzmetal angina)

Atherosclerotic
- 1) Stable Angina
- 2) Unstable angina
- 3) Myocardial Infraction

Acute Coronary Syndrome ← 2), 3)

1) Angina Pectoris
   - ↳ Chest
   - "To strangle"

→ Chest pain due to reduced Blood flow.

1) Stable angina → Most common type.
   - → 70% stenosis, but other 30% open.
   - → No pain in rest, but severe pain when at exercise or stress, because heart needs more blood.

Cause
- → i) Atherosclerosis → Most common
- ii) others → Hypertrophic cardiomyopathy, Aortic stenosis, Hypertension.

Pathology
- → Blood flow Cut off to Subendocardium
  - ↓
  - Release of adenosine, Bradykinin, others.
  - ↓
  - Pain to, left arm, jaw, shoulder or back. Sweating, Shortness of Breath -

→ Pain usually for 20 minutes, and goes away after rest.

2). Unstable angina

→ Pain occurs during @ Rest or stress or at rest and does not go away in 20 minutes with plaque rupture.

→ If not treatment early then might progress for Myocardial ischemia (Necrosis started) and Angina (heart tissue alive but Ischemic).

→ Have Subendocardium blood infract causes pain.

3). Prinzmetal angina (Vasospastic angina).

→ May or may not have atherosclerosis and might occurs any time, At rest also.

→ Cause → Vasospasm sudden
↓
Transmural ischemia

Stable & Unstable = ST segement depression.
↳ Subendocardial ischemia.

Prinzmetal angina = ST segment elevation.

Clinical Features

S - Diffuse, difficult to localize, Substernal.
O - Sudden, rest, exercising.
C - Pressure, Heaviness, Tightness, Squeezing.
R - To Neck, Jaw, arm, Back.
A - ↓ Position, Respiration, motion,
T: ↑
E → Sweat, Vomiting, Bradycardia, Hypotension.
S - Severe.

First 10 Minutes → Examination

1) Check A → Airway
   B → Breathing
   C → Circulation.
2) Physical examination.
3) Oxygen Saturation.
   < 90% → Supplement oxygen.
   < 80% → Supplemental ventilation oxygen.
4) Resuscitation equipement nearby.
   → Defibrillator.
   → Airway Equipement.
5) 325 mg Aspirin (Blood thinner).
   ↳ Unless contradiction (Anaphylaxis or already given.

Investigation:

1) Blood →
   ↳ CBC, Electrolytes.
   → Creatinine, BUN, Coagulation factors.
   → Lipids.
2) Cardiac Biomarkers → Acute myocardial damage
   → Troponin T & I → Due to destruction of Cardiomyocytes.
   → Positive in Myocardial infarction and some cases of prinzmetal angina.
   → Creatinine Phosphokinase and LDH are non sensitive and less specific.
   → Brain natriuretic peptide.
3) 12 lead ECG → For 24 hours
   ↳ STEMI → Prinzmetal angina & Myocardial infarction
   → NSTEMI → Subendocardial infract.

④ Stress test :-

→ Measure heart ability to respond to external stress in controlled clinical environment

Treatment

① Nitrates → Widens arteries, ↑ Blood flow to heart

In prinzmetal can be diagnosis

↳ 3 Sublingual Doses of 0.4mg, 1 every 5mins -

② β - Blockers :-

⇒ Constricts the vessels and reduces the cardiac demand.

⇒ Contraindicated in prinzmetal.

Coronary Artery Disease.

Left Coronary Artery — Anterior descending → Anterior
Circumflex → lateral wall

Right Coronary artery - Posterior wall

Etiology:

i) Atherosclerosis
ii) Non-Atherosclerotic → Arteritis. Spasm of artery. Embolism, Trauma, Thrombosis, Thickening. Aneurysm

i) Atherosclerosis:

Unmodifiable risk factor:
- Age - Higher
- Family history
Best - Male, Female after menopause
Personality who are aggressive

Modifiable:
- Cigarette smoking
- Sedentary life style
- Hyperlipidemia ↑HDL, ↑LDL
- Hypertension.
- Diabetes mellitus.

Pathology:

→ ↑↑ Endothelial damage
↓
↑LDL inside Endothelial
↓ Immune response
Macrophages engulf LDL
↓ Becomes
Dead Monocyte (Foam cells)
↓ More cytokines.
Fatty Streak.
↓ ↑ Platelet derive Growth factor
↑ Smooth Cell Proliferation
↓
Forms Fibrous Cap + Fatty Streak
↓ Becomes plaque

Occluded artery

Coronary → Angina + MI

Cerebral → Stroke + Cerebral atrophy

Superior mesentric - Small intestine

Popliteal → Gangrene + claudication

Renal → Renin. angiotensin. Hypertension.

Nomenclature

Type (I) initial lesion (Isolated macrophage / foam cells.

Type II Fatty streak - (Mainly Intracellular lipid accumulation)

Type III (Intermediate) lesion (Type II + Small extracellular lipid pools)

Type IV (Atheroma) lesion (Type II + confluent extracellular lipid).

Type V (Fibroatheromatous lesion. (Lipid core + Fibrous layer

Type VI (Complicated lesion (Haematoma lesion)

Clinical Presentation:

- Acute MI → STEMI + NSTEMI
- Unstable angina
- Stable angina
- Arrhythmia.
- Cardiomyopathy.
- Sudden cardiac death

Acute Coronary Syndrome:

i) Myocardial Infraction
ii) Unstable Angina

i) Myocardial Infraction:

- Death of the myocardial tissue due to lack of oxygen rich blood flow.

Risk Factors:- Same Atherosclerosis

Pathogenesis:- Same "

- When it affects the endocardium and myocardium.

1 minute - Reversible.

Typs 20-40 minute → Irreversible.

When Blood comes then it is called Subendocardial infract.

NSTEMI.

When Blood does not come whole layers affected called as Transmural infract (STEMI). (2hrs).

Clinical features

Symptoms:

- Chest pain radiating to left arm or jaw
- Sweating. Nausea, Vomiting. Dyspnea
- Painless in diabetes Mellitus.

Signs:

i) Sympathetic activation → Tachycardia, sweating pallor
ii) Vagal → vomiting → Bradycardia
iii) Impaired myocardial function → Hypotension, oliguria. Cold extremities. Narrow pulse pressure, Crepitation. Fourth heart sound.
iv) Tissue damage → Fever.

Investigations

- Cardiac enzymes:

  Troponin T & Troponin I → 2-4 hrs after, peaking in 1st wk after 7-10 days, 2 wks.

  CK-MB → 4 hrs, 24 hrs, return after 4 days.

- ECG:

within 30 minutes - ST elevation - STEMI Transmural
ST depression/No elevation - NSTEMI - Subendocardial

Before 24 hrs - T wave inversion

After 24 hrs - Q wave disappears.

- ↑ C Reactive protein, ↑ ESR, ↑ Leucocytes

→ Chest X ray → Normal in acute. Cardiomegaly in massive attack.

→ Echocardiography:

Complications:

0 - 24 hrs → Arrhythmia, Cardiogenic shock

1 - 3 days → ↑ Neutrophils due to inflammation
Pericarditis.

3-14 days → ↑ Granulation tissue. → Myocardial rupture

2 weeks → Scarring → Heart failure

Treatment:

- Fibrinolytic therapy
- Angioplasty
- PCI Stent - Antiplatelets, Anticoagulants, Nitrates
- Beta blockers, Pain medications